Study mortality with hazard rates, not probabilities

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Running headline: Mortality hazard rates and probabilities

Abstract

1. Mortality is a key process in ecology and evolution, and much effort is spent on statistical and theoretical modelling of this process. Mortality hazard rates describe individuals' instantaneous ability to survive at different times, whereas survival probabilities are defined for time-intervals of a given length. We argue that it is often more informative to model and interpret time-averaged mortality hazard rates in cases where survival probabilities are traditionally used.
2. We discuss three areas where modelling of survival probabilities may be little informative or even misleading, and where mortality hazard rates are more suited for inference; (i) modelling of covariate effects on survival probabilities versus mortality hazard rates, (ii) modelling of multiple sources of mortality and competing risks, and (iii) elasticity analyses of population growth rate with respect to survival probabilities versus mortality rates.
3. By focusing on modelling (time-averaged) mortality hazard rates (measurements on a ratio scale), one avoids several inherent problems of comparing survival or mortality probabilities (measurements on an absolute scale). In particular, statements about relative differences between mortality hazard rates, or effects of relative changes in mortality rates on population growth (elasticities), are more informative than similar statements involving survival probabilities.
4. The concept of hazard rates is essential for understanding ecological and evolutionary processes and we provide an intuitive explanation for this, using several examples.

Keywords: Capture-mark-recapture, time-to-event data, cause specific mortality, competing risks, measurement theory, time-averaged hazard rates, loglog link function, odds-ratio vs. hazard-ratio.

Introduction

Inferences about demographic processes in ecology are commonly based on capture-recapture studies where individuals are observed at fixed time-intervals (McCrea & Morgan 2015). **Survival probability** over such an interval from time t_1 to time t_2 ($S_{t_1 \to t_2}$) is a function of **mortality hazard rate** ($h(t)$),

$$S_{t_1 \to t_2} = e^{-\int_{t_1}^{t_2} h(t)dt} \tag{1}$$

(Box 1; terms in bold at first occurrence are defined in the Glossary). By taking the natural logarithm of both sides of this equation and dividing by the length of the interval, we obtain the time-averaged mortality hazard rate for the interval,

$$\bar{h}_{t_1 \to t_2} = \frac{\int_{t_1}^{t_2} h(t)dt}{t_2 - t_1} = -\frac{\log\left(S_{t_1 \to t_2}\right)}{t_2 - t_1} \tag{2}$$

(Collett 2014). Such interval specific survival probabilities and time-averaged hazard rates are often sufficient for specific analyses where fluctuations within intervals (e.g. due to seasonal or daily cycles) are not relevant.

Although the time-averaged mortality hazard rate is a simple transformation of survival probability (eq. (2)), there are some important differences in how these two quantities (measurements) can be interpreted and modelled. The survival probability is a unit-free measurement on an **absolute scale**, meaning that no transformation can be performed without changing its *meaning* (Houle *et al.* 2011). For example, if $S = S_{t_1 \to t_2}$ is the probability of surviving a whole year, then $S^{1/2}$ can be interpreted as the probability of surviving half the year, assuming that survival probabilities in the first and second half of the year are equal. The mortality hazard rate, on the other hand, is a **ratio-scaled measurement**, where unit of measurement may be changed (e.g., from ‘year^{-1}’ to ‘month^{-1}’) by simply multiplying with a positive number (just like a given distance can be measured in ‘meters’ or ‘feet’) (Houle *et al.* 2011).

In capture-recapture studies, structured population modelling and life-history theory, the survival process is usually represented by survival probabilities over fixed time or age intervals. In fishery

studies, however, it is more common to use mortality hazard rates in the population dynamical models. Sometimes the choice of using survival probabilities versus mortality hazard rates is simply a matter of interpretation/presentation or alternative parameterizations of the same model. However, in hierarchical models where interval (and individual) specific survival probabilities are constrained as functions of covariates, the choice of measurement can have important consequences for the analysis. Nevertheless, the rationale for this choice is seldom specified. In general, there appears to be little contemplation about how survival probabilities relate to the processes of interest in the ecological literature. We will here discuss three important areas where modelling of survival probabilities may be little informative or even misleading, and where mortality hazard rates are more suited for inference; (i) modelling of covariate effects on survival probabilities versus mortality hazard rates, (ii) modelling of multiple sources of mortality and competing risks, and (iii) elasticity analyses of population growth rate with respect to survival probabilities versus mortality rates.

Logit-linear models of survival vs. log-linear models of mortality rates

When modelling survival probabilities, effects of covariates are typically incorporated through link-functions, such as the **logit-link** or the **loglog-link**, guaranteeing probability values between 0 and 1 within a generalised linear modelling framework. When a logit-link function is used for survival probabilities, the exponent of **linear contrasts** may be interpreted as **odds-ratios**, whereas, when a loglog-link is used, the exponent of linear contrasts becomes **hazard ratios** (Box 1). In other words, when using a logit-link, it is the **relative difference** in survival **odds** that is modelled, while using a loglog-link implies relative effects on mortality hazard rates (Box 1).

While most practitioners are presumably aware of the different functional forms implied by the choice of link-function when using continuous covariates, there seem to be less awareness of the fact that parameterising the models with survival probabilities representing a different duration (e.g. weeks instead of months) also changes the functional forms when using any other link-function than the loglog-link (Figure 1). That is, when *not* using a loglog-link for survival probabilities, selecting the time-unit for the analysis (which can be set in commonly used computer programs such as MARK (White 2016)) is part of the model definition. In many studies, it is natural to model survival on an annual time scale as this represents survival over a full seasonal cycle. However, for species with a life-expectancy of much less than a year, such as many small rodents, yearly survival has little meaning, and one may choose to model weekly or monthly survival instead. Sometimes it may also be of interest to estimate the effects of certain covariates on survival until a specific life-stage (e.g. the effect of chick body mass on survival until reproduction in a long-lived seabird). If a logit-link is used in such cases, one will get different results depending the choice of time unit that the survival probabilities in the model represent. Using a loglog-link for survival, which implies a log-linear model for the time-averaged mortality hazard rate (Box 1), avoids the problem of selecting the time-spans

that the survival probabilities represent because survival over any given interval is then invariant to the interval length used in the modelling of covariate effects (Figure 1). One can then instead focus on developing a good model for the mortality hazard rate (Ergon, Yoccoz & Nichols 2009; Choquet *et al.* 2017).

While survival probabilities usually provide meaningful inference, differences in survival expressed as odd-ratios are often difficult to interpret in a biological context. For example, if yearly juvenile survival is 0.25 (odds = 1/3) and yearly adult survival is 0.50 (odds = 1), the odds of surviving one year is 3 times higher for adults compared to juveniles, but the odds of surviving two years (which may correspond to the age at maturation) is 5 times higher for adults than juveniles (1/3 vs. 1/15). If the environment improves such that yearly survival of juveniles and adults increases to 0.50 and 0.71 respectively, the odds ratios for surviving respectively one or two years is now 2.4 and 3.0, suggesting a smaller difference between juveniles and adults in this environment. In both of these examples, however, the time-averaged mortality hazard rate (eq. 2) for juveniles is 2 times higher than the mortality hazard rate for adults, meaning that the intensity at which deadly events occur is twice as high for juveniles compared to adults, irrespective of the environment and the time-scale used. Such hazard ratios thus have a clear biological interpretation, and facilitate meaningful comparisons of effect sizes across studies. As illustrated in Figure 2a, odds-ratio for any given hazard ratio depend on the length of the time-interval that the odds apply to, as well as the survival probability of the reference group. The choice of link-function also dictates the meaning of model constraints, and choosing biologically reasonable constraints can improve model fits.

Multiple sources of mortality and competing risks

Discerning between different sources of mortality is of prime importance in ecology, evolutionary biology and population management (Schaub & Lebreton 2004; Péron 2013; Wolfe *et al.* 2015). To understand selective forces and population dynamics, it is also important to examine how different sources of mortality co-vary and interact (Nichols *et al.* 1984; Boyce, Sinclair & White 1999; Koons *et al.* 2014). For example, an essential question is whether increased hunting or harvesting will lead to a reduction (or potentially an increase) in mortality due to other causes. Such effects can result from density dependent responses or non-random removal of weaker (or stronger) individuals (i.e., selection) in a heterogeneous population. Changes in one source of mortality may also lead to evolutionary changes in other mortality rates or life-history traits. For example, age-dependent mortality patterns affect optimal investments in development and maintenance/longevity as well as reproductive effort at different ages (Charlesworth 1980; Caswell 2007a; Koons *et al.* 2014).

When studying multiple sources of mortality, it is essential to realise that if the hazard rate representing one cause of mortality (say, hunting) increases, the probability of dying from any other cause will automatically be reduced, even if the mortality hazard rates representing the other causes

remain unchanged. This is simply because it has now become more likely that the individual will die of this cause (hunting) before other deadly events occur to the individual (Box S1, Supporting Information). We may say that the different causes of mortality "compete" over being the one that kills the individual first, and the probability of "winning" depends on how "strong" the competitors are (referred to as "competing risks" in survival analysis (Collett 2014)).

If we assume that there are K causes of mortality and the mortality hazard rates for each cause k, $h_k(t)$, remain proportional within a time interval, such that the fraction $\alpha_k = h_k(t)/\sum_{l=1}^{K} h_l(t)$ remains constant for all causes throughout the interval, it can be shown (see Box S1) that the probability of dying from cause k (i.e., the '**risk**' of cause k) is

$$P_k = (1 - S)\alpha_k \,, \tag{3}$$

where S is the probability of surviving the interval (from t_1 to t_2) defined to have length 1. That is,

$$S = e^{-\int_{t_1}^{t_2} \sum_{k=1}^{K} h_k(t)dt} = e^{-\sum_{k=1}^{K} \int_{t_1}^{t_2} h_k(t)dt} = e^{-\sum_{k=1}^{K} \bar{h}_k} \,, \tag{4}$$

where $\bar{h}_k$ is the time-averaged hazard rate for cause k (eq. (2)). These parameters would in the most general case be interval- and individual-specific, but indices for interval and individual are omitted here for simplicity. Note that when the hazard rates $h_k(t)$ remain proportional within an interval, the relative differences between them are the same as the relative differences between the time-averaged hazard rates. Hence, we can also interpret α_k as

$$\alpha_k = \frac{\bar{h}_k}{\sum_{l=1}^{K} \bar{h}_l} \,. \tag{5}$$

When information about cause of death (e.g., harvesting) is available for at least a subset of individuals in the population, multi-state capture-recapture models can be fitted to the data to address questions relating to e.g. compensatory effects of harvesting or age-related patterns in mortality from different causes (Servanty *et al.* 2010; Koons *et al.* 2014). Such models involve probabilities of remaining in an "alive-state" (survival probability S) or transitioning to any of the absorbing death-by-cause states (probabilities P_k's). From equations (3), (4) and (5) we see that there are at least three

distinct ways to parameterize these transition probabilities when hazard rates within intervals are assumed to be proportional: [1] with S and P_k's under the constraint that $S + \sum_{k=1}^{K} P_k = 1$; [2] with S and α_k's under the constraint that $\sum_{k=1}^{K} \alpha_k = 1$; or [3] with the cause-specific time-averaged mortality hazard rates $\bar{h}_k$'s. The relationships between the parameters in these three parameterizations are given in Table 1. Note that when constraining these individual- and interval-specific parameters by e.g. functions of individual age, these alternative model formulations will generally lead to different models.

Since a cause specific **mortality probability**, P_k, does not only depend on the mortality hazard rate for this cause, but also on the total mortality hazard rate (Box S1), it will in general be more biologically interesting to model the cause specific mortality hazard rates (h_k's). Hazard rates describe the fundamental stochastic process of mortality (Box 1), and can be modelled as functions of environmental or individual covariates, or by the use of correlated random effects across years. Nevertheless, statistical modelling of capture recapture data incorporating multiple (usually two) sources of mortality, has so far mostly been based on the mortality probabilities ($P_k's$) (references in Table 1), perhaps because these can be estimated and modelled through general multi-state capture-recapture models (Lebreton *et al.* 2009) as implemented in commonly used programs such as MARK (White 2016) and E-SURGE (Choquet, Rouan & Pradel 2009). The majority of ecological studies of multiple mortality causes using time-to-event data from telemetry or loggers (Heisey & Patterson 2006) also put undue emphasis on mortality probabilities rather than hazard rates, sometimes leading to misguided conclusions.

Many studies have estimated relationships between probabilities of dying from different causes across years or space in order to assess whether mortality from these causes are additive (not associated), compensatory (negatively associated) or depensatory (positively associated) (Bischof *et al.* 2009; Servanty *et al.* 2010; Péron 2013; Wolfe *et al.* 2015). One problem of using mortality probabilities in such studies is that they are, as explained above, intrinsically related; increasing the mortality hazard rate of one cause implies that the probability of dying from this mortality cause will increase, while the probability of dying from all other causes decreases (Box S1). As shown in Figure 3, this intrinsic negative association may dominate empirical correlations between cause specific mortality probabilities across years (or groups of individuals) even when correlations between the cause specific mortality hazard rates across years (or groups) are strongly positive. Realizing this problem, researchers have come up with innovative ways to reduce "bias"[1] in correlations between cause-specific mortality probabilities (Schaub & Lebreton 2004; Servanty *et al.* 2010) or to interpret these correlations with respect to hypotheses for compensatory versus additive mortality patterns

[1] Note that correlations between mortality probabilities only are "biased" in the sense they do not represent correlations between cause specific mortality rates. They are not biased when interpreted strictly as correlations between interval-specific mortality probabilities.

(Péron 2013). However, when modelling mortality hazard rates, no such corrections are necessary to obtain the desired interpretation; when partitioning mortality into one specific cause (e.g. "harvesting/predation") and all other causes, a negative correlation between the mortality hazard rates among years indicates compensation, a positive correlation indicates depensation, and a correlation close to zero may indicate additivity. Furthermore, a near constant sum of the hazard rates indicates perfect compensation, while a negative correlation between the sum of the hazard rates and the harvesting hazard rate indicates overcompensation. When assessing such correlations it is important to ensure that the process correlations are not confounded with sampling correlations, for example by using appropriate hierarchical models (Royle & Dorazio 2008).

In a multi-state capture-recapture model with two competing causes of mortality, Schaub and Pradel (2004) parameterized the transition probabilities from an 'alive' state to the two cause-specific 'dead' states as $P_1 = (1 - S)\alpha$ and $P_2 = (1 - S)(1 - \alpha)$. Here, $(1 - S)$ is the probability of dying from any cause, and α was defined as the probability of having died from cause 1 given that the individual has died. Note that, as can be seen from equation (3) above, α can also be interpreted as the proportion of the total mortality hazard rate attributable to cause 1 under the assumption that the hazard rates for the two mortality causes remain proportional within the interval (i.e., the second parameterization in Table 1). As such, α and S are more revealing about the underlying processes than P_1 and P_2, and it may in some applications make more sense to model individual or time-specific α and S as a function of covariates than similar models for the P's. To investigate interactions between causes of mortality, it is however necessary to estimate correlations between $\bar{h}_1 = -\log(S)\,\alpha$ and $\bar{h}_2 = -\log(S)\,(1 - \alpha)$ across intervals or groups of individuals.

Our recommendation to model and interpret mortality hazard rates instead of mortality probabilities is also highly relevant for studies of age related patterns in mortality. For instance, Koons *et al.* (2014) assessed patterns of senescence in a population of snow geese (*Chen caerulescens*) and a population of roe deer (*Capreolus capreolus*) by estimating age-dependent cause specific mortality probabilities pertaining to hunting/human-related causes as well as natural causes. For both species they concluded that the yearly probability of dying from hunting/human-related causes was nearly independent of age, while the probability of dying from natural causes increased sharply at higher ages. However, as explained above, a model assuming that the probability of dying from human-related causes is constant, while the probability of dying from non-human related causes increases with age, implies that also the mortality hazard rate representing human related causes must increase (Figure 4). In empirical studies of senescence, one should also consider potential biases arising from un-modelled individual heterogeneity (i.e., "**frailty**") (Vaupel, Manten & Stallard 1979; Cam *et al.* 2002; Collett 2014), temporary emigration (Langtimm 2009) and age-dependent dispersal (Ergon & Gardner 2014).

Lastly, we note that theoretical analyses using population projection matrices (Caswell 2001) or projection kernels for continuous states (Easterling, Ellner & Dixon 2000; Ellner, Childs & Reeds

2016) often involve stochastic transitions to several alternative states (e.g., reproductive states and death). When these transitions can take place at any point in time within the intervals (e.g. infections and mortality), an increase in the hazard rate for one transition will reduce the probability of the other (competing) transitions even if the hazard rates for these transitions remain the same. Hence, we recommend parameterising also such theoretical models by the use of transition specific hazard rates instead of transition probabilities (see Box S1 and Table 1).

Analyses of elasticity of population growth rate to survival versus mortality rates

Sensitivity analyses are essential in studies of population dynamics, management of natural populations, life-history theory and evolutionary modelling (Benton & Grant 1999; van Tienderen 2000; Caswell 2001; Caswell 2007b). In particular, many studies consider the sensitivity of the asymptotic population growth rate (λ) to changes in demographic parameters such as survival probability (S) and fecundity (F), as well as lower-order parameters and environmental variables. It is common to interpret and compare such sensitivities with respect to different demographic parameters on a relative (logarithmic) scale. That is, the relative change in growth rate per relative change in, for example, survival during one life-stage, referred to as the *elasticity* of λ to survival $\left(\epsilon_S = \frac{\partial \log \lambda}{\partial \log S}\right)$, is compared to the elasticity of λ to fecundity $\left(\epsilon_F = \frac{\partial \log \lambda}{\partial \log F}\right)$ (Caswell 2001). However, this comparison is implicitly dependent on the length of the time period that the survival probability applies to (often set to one year). For example, instead of calculating elasticities to yearly survival probability (S_y) during the pre-reproductive life-stage, one could calculate elasticities to survival over two years (which may correspond to age of maturation). If we, for simplicity, assume that survival probability through the first and second year are the same, survival over the two years becomes $S_{2y} = S_y^2$. Population growth rate is naturally more sensitive to a relative increase in S_y than to the same relative increase in S_{2y}; elasticity of λ to S_y is twice the elasticity of λ to S_{2y} ($\epsilon_{S_y} = \frac{\partial \log \lambda}{\partial \log S_y} = \frac{\partial \log \lambda}{\partial \log S_{2y}} \frac{\partial \log S_{2y}}{\partial \log S_y} = 2\frac{\partial \log \lambda}{\partial \log S_{2y}} = 2\epsilon_{S_{2y}}$). Now consider instead the elasticity of λ to the time-averaged mortality hazard rate, $\bar{h} = -\log(S)$, which is $\epsilon_{\bar{h}} = \log S \frac{\partial \log \lambda}{\partial \log S} = -\bar{h}\epsilon_S$. This elasticity is invariant to any re-parameterizations on the form $S_{2y} = S_y^2$ because $\log S_{2y} \frac{\partial \log \lambda}{\partial \log S_{2y}} = \log S_y \frac{\partial \log \lambda}{\partial \log S_y}$.

Figure 5 shows these relationships between elasticities of λ to survival probability and mortality hazard rate when the model is parameterized with survival probabilities over different time intervals. Clearly, if the purpose of the elasticity analysis is to investigate the effect of a relative change in the ability to survive during an interval of a somewhat arbitrary length, it is more appropriate to base the elasticity analysis on the time-averaged mortality hazard rate ($\bar{h}$) than on the probability of surviving

this interval (S). Note also that elasticity of λ to survival probability (S) does *not* equal the negative elasticity of λ to probability of mortality ($1 - S$) (Link & Doherty 2002). Moreover, a given environmental or genetic change is not likely to have the same relative effect on a high vs. low survival probability (e.g., a 10% increase in survival could represent a change from 0.10 to 0.11 or from 0.90 to 0.99, and a 10% increase is impossible when survival probability is 0.95). Instead, it seems generally more plausible that a certain environmental change, management action or genetic change, will have a proportional effect on the intensity at which deadly events occur; i.e., the mortality hazard rate (see Box 1).

One motivation for using elasticity analysis (i.e., analysing relative effects of relative changes) instead of sensitivity analysis of **absolute effects** to absolute changes, is that a unit change in transition probabilities such as survival and maturation probabilities is not comparable with a unit change in fecundity (Caswell 2001, p. 226; Link & Doherty 2002). However, it can be argued that also a comparison between the effects of relative changes in a variable on an absolute measurement scale (transition probabilities taking values in the [0,1]-range) and a ratio scaled variable (fecundity) is not meaningful (Houle *et al.* 2011). It seems much more meaningful to compare relative effects of relative changes (i.e., elasticities) in two ratio-scaled variables such as mortality hazard rate and fecundity.

In the example used in Figure 5a, where yearly juvenile survival probability is much lower than yearly adult survival probability, population growth rate has a higher elasticity to adult survival than to juvenile survival. This pattern is typical of long-lived vertebrates, and is commonly interpreted as population growth rate and fitness being more sensitive to survival of adults than juveniles in such populations (see e.g. references in Gaillard and Yoccoz (2003)). However, elasticity with respect to mortality hazard rates shows the *opposite* pattern; elasticity to juvenile mortality rate is higher than to adult mortality rate. These counterintuitive differences in elasticities to survival probability and mortality rate do not occur when juvenile survival and fecundity is sufficiently high compared to adult survival (Figure 5b). Figure 6a shows how elasticities of λ with respect to survival probabilities relate to juvenile and adult survival, and Figure 6b shows the equivalent relationship for elasticities of λ with respect to mortality hazard rates. These very different patterns emerging from elasticity comparisons with respect to survival and mortality rate are due to the fact that a relative change in mortality hazard rate leads to a small relative change in survival probability when survival is high, but a much larger relative change when survival probability is low (Figure 7). Clearly, an uncritical comparison between elasticities to survival probabilities at different life-stages may lead to misguided inference.

Sensitivities also play an important role in our understanding of underlying mechanisms to population growth and extinction risk in fluctuating environments. Several studies have compared sensitivities of the average population growth rate (or fitness) to demographic parameters in relation to their variability over time (reviewed in McDonald *et al.* (2017)). Most analyses conclude that life-history traits to which fitness is most sensitive tend to be more buffered (i.e., canalised) against

environmental fluctuations than other traits. However, some authors have pointed out that such conclusions may be highly dependent on the measurement scale of the life-history traits (references in McDonald *et al.* (2017)). In particular, there has been a concern that among-year variance in survival and state transition probabilities are bounded by a maximum attainable variance given by their mean, and hence cannot be as variable as other traits (Link & Doherty 2002). Using variance-stabilising transformations as advocated by Link and Doherty (2002), McDonald *et al.* (2017) reported on an analysis of demographic strategies in a wide range of plants and found that logit-transformed survival probabilities were in fact often highly labile (i.e. not canalised) despite strong variance-standardised sensitivities (VVS_q) for these traits (VVS_q is here defined as $\mathrm{VVS_q}(\lambda, \mathrm{S}) = \mathrm{Sensitivity}(\log(\lambda), q(S))$ where $q(S)$ is variance-standardised transformation of survival; Link and Doherty 2002). The use of the logit function as a variance-stabilising transformation of survival probabilities in this analysis assumes that among-year variance in survival probability is proportional to $\mu_S^2(1-\mu_S)^2$ where μ_S is the mean survival among years. Alternatively, the same analysis could have used a loglog-transformation of survival probabilities which would stabilize the variance when the variance in survival probability is proportional to ${\mu_S}^2 \log(\mu_S)^2$. Since among-year variability in survival probability is presumably highly system specific as it depends on complex trophic interactions and (non-linear) environmental effects relating to for example predators, pathogens, climate and intraspecific population density, it is difficult to argue that one variance-mean relationship is generally more applicable than others. We also note that among-year variance in survival probability seems to typically be far below the maximum attainable value of $\mu_S(1-\mu_S)$ (see data in Gaillard and Yoccoz (2003) and McDonald *et al.* (2017)). Hence, we do not advocate the use of loglog-transformation of survival probabilities due to its particular variance-stabilizing properties, but instead because this transformation is equivalent to a log-transformation of mortality hazard rates (Box 1). This means that the variance-stabilized sensitivity ($\mathrm{VVS_q}(\lambda, \mathrm{S})$) for survival using a loglog-transformation ($q(S) = \log(-\log(S))$) is identical to the elasticity for mortality hazard rate ($\mathrm{VVS_q}(\lambda, \mathrm{S}) = \epsilon_{\bar{h}}$). As argued above, it is more meaningful to compare elasticities of two ratio-scaled variables such as mortality hazard rate and fecundity than relying on some rather arbitrary transformations of survival probabilities for such comparisons.

Authors' contributions

TE conceived the main ideas and wrote the first version of the manuscript. All authors contributed with discussions, literature review, commenting and editing of subsequent versions.

Box 1 – Mortality hazard rates and survival probabilities

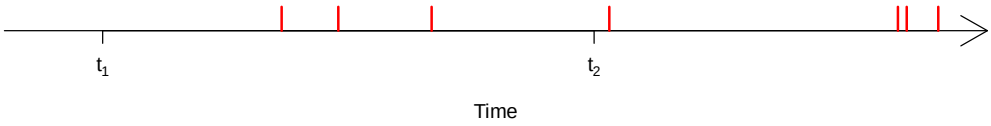

Figure B1. Realized times of recurrent events (or "deadly events experienced by an imortal individual")

A mortality hazard rate is the underlying intensity of deadly events that an individual is exposed to. To better understand the relationship between survival probabilities and hazard rates, first consider a point process of non-lethal events (*e.g.*, ingestion of food items). An example of realized times of such events for an individual is shown in Figure B1. Denoting the intensity of this point process as a function of time as $\lambda(t)$, the expected number of events occurring during an interval from time t_1 to time t_2 is the integrated intensity over this interval, $\int_{t_1}^{t_2} \lambda(t)dt$. When events occur independently of each other, the number of events X occurring in the interval is a random Poisson variable, $X \sim Poisson\left(\int_{t_1}^{t_2} \lambda(t)dt\right)$. If these events are lethal, X must be zero for the individual to survive the interval, which according to the Poisson distribution occurs with probability $S_{t_1 \to t_2} = \Pr(X = 0) = e^{-\int_{t_1}^{t_2} \lambda(t)dt}$. When studying lethal events, the event intensity is called a 'hazard rate' and is often denoted as $h(t)$ ($= \lambda(t)$ above). If the hazard rate is considered as constant over an interval of length $\Delta t = t_2 - t_1$, survival probability over this interval becomes $S = e^{-h\Delta t}$. When the hazard rate is not constant over the interval, the relationship $S = e^{-\bar{h}\Delta t}$ still holds when parameter $\bar{h}$ is interpreted as a 'hazard rate average' (Collett 2014).

Mortality hazard rates may thus be interpreted as the "expected number of deadly events occurring per time-unit for an immortal individual". Since hazard rates are **ratio scaled** intensities, they may be expressed with an arbitrary time-unit and values are proportional to the length of the time-unit used (e.g, a hazard rate of 1 month^{-1} is equivalent to 12 year^{-1}). The ratio of two hazard rates, a **hazard ratio**, is thus invariant to the time-unit used to express the rates and has a clear interpretation; if the mortality hazard rate for juveniles is twice the mortality hazard rate of adults, we know that juveniles are exposed to twice as many expected deadly events per time-unit than adults. Such hazard ratios may be estimated by modelling survival probability through a **loglog-link**; $S = \exp(-\exp(\eta))$, where η is the linear predictor and $\exp(\eta)$ is the time-averaged mortality hazard rate over the interval, expressed with the interval length as a time-unit. If survival probability of one group of individuals is

represented by $\eta_1 = \beta_0 + \beta_2 \log(x)$ and another by $\eta_2 = \beta_0 + \beta_1 + \beta_2 \log(x)$, where x is a continuous covariate, the hazard ratio representing their **relative difference** in mortality, irrespective of the covariate, becomes $\frac{\exp(\eta_2)}{\exp(\eta_1)} = \exp(\beta_1)$. Further, when the covariate is multiplied by a factor C, the mortality hazard rate will be multiplied by a factor C^{β_2}(when $\beta_2 = 1$ the hazard rate is proportional to the covariate). Hence, when using a loglog-link on survival probabilities we model relative differences in hazard rates (i.e., hazard ratios). [Note that some computer programs like MARK (White 2016) define the loglog-link function as $S = \exp(-\exp(-\eta))$, in which case the above hazard ratio becomes $\exp(-\beta_1)$.]

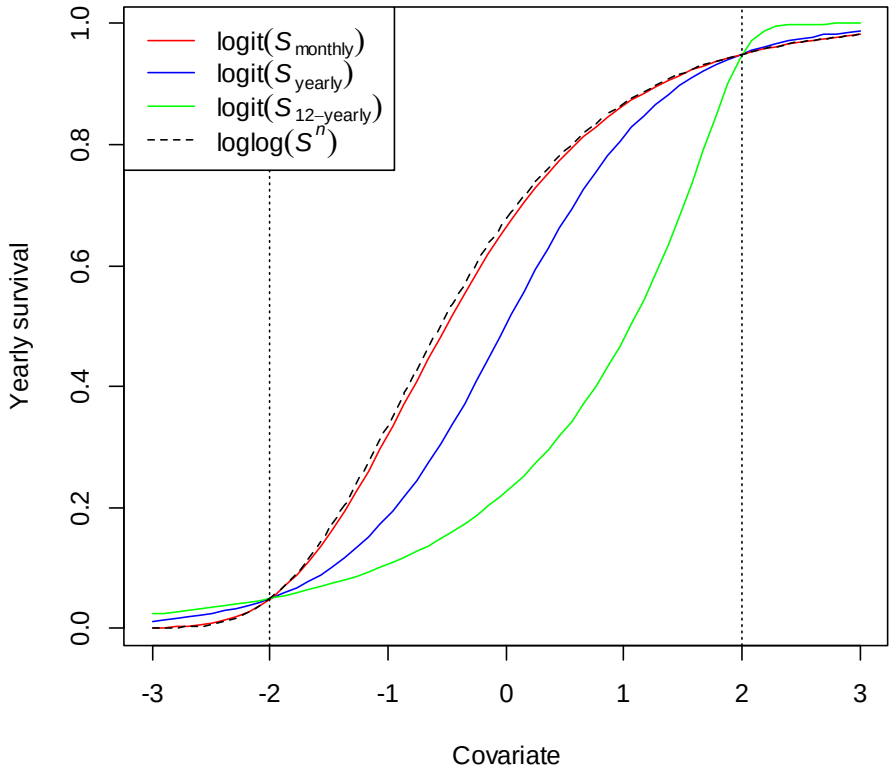

Figure 1. Functional shape of the relationship between yearly survival and a covariate when using the logit link-function on survival probability over different time interval lengths (solid coloured lines; see legend). When the logit-link is applied to monthly survival (red line), it is assumed that probability of survival is the same in all of the 12 months of the year, such that $S_{\text{yearly}} = S_{\text{monthly}}^{12}$. Similarly, when the logit-link is applied to survival over 12 years, $S_{\text{yearly}} = S_{12-\text{yearly}}^{1/12}$ (green line). To facilitate comparison of functional shapes, slope and intercept parameters are defined such that yearly survival probability is 0.05 at covariate value -2 and 0.95 at covariate value 2. The dashed line shows the loglog-linear relationship between yearly survival and the covariate, which is invariant to the interval length for the survival probability. When the logit-linear relationship applies to survival probabilities over short intervals, it approaches the loglog-linear functional form (see red line).

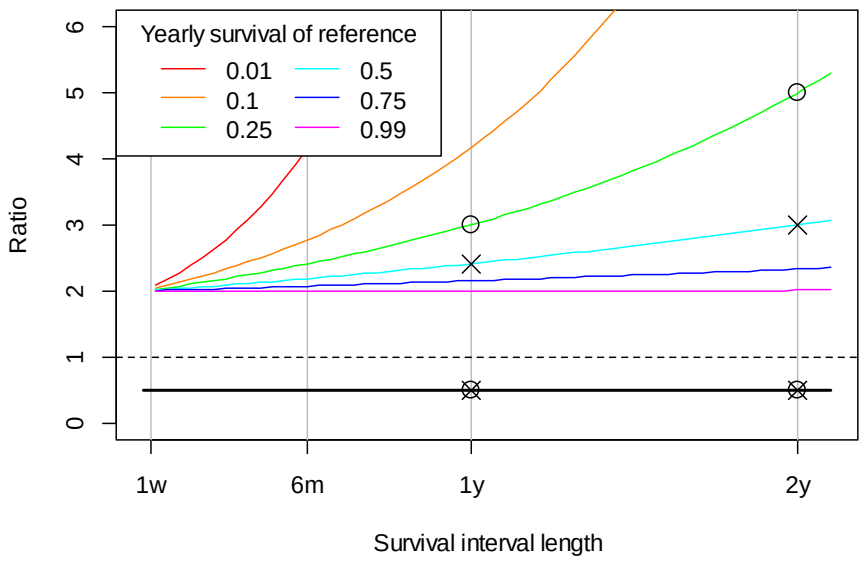

Figure 2. Contrasts between two survival probabilities expressed as odds-ratios as a function of time interval length for survival (x-axis), when the mortality hazard ratio is 0.5 (indicated with thick black lines) and yearly survival probability of the reference varies according to the colour code in the legend. Plotted points refer to the example in the text (circles represent the original environment, while crosses represent the improved environment). Vertical lines are plotted at 1 week, 6 months, 1 year and 2 years.

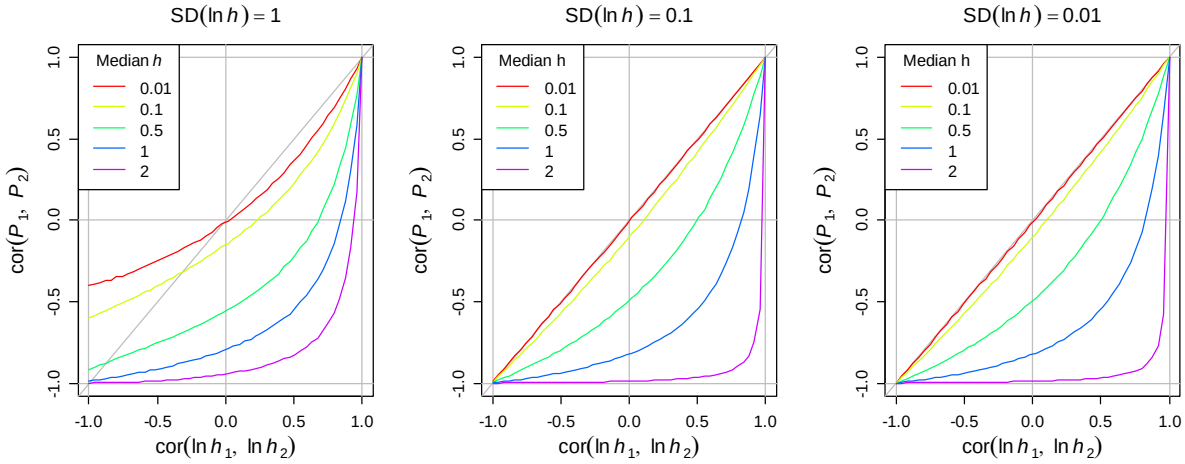

Figure 3. Correlations between probabilities of two mutually exclusive and collectively exhaustive causes of mortality (e.g. among years) as a function of the underlying correlation between the corresponding mortality hazard rates on a logarithmic scale (x-axes), assuming that the two hazard rates h_1 and h_2 have a bivariate log-normal distribution. Correlations were computed by first drawing 10^5 values of $\ln(h_1)$ and $\ln(h_2)$ from the bivariate normal distribution and then computing the Pearson correlation coefficients of P_1 and P_2 (Table 1). The standard deviation of $\ln(h_1)$ and $\ln(h_2)$ varies by panel as indicated above the plots. Different lines represent different median hazard rates according to colour legend.

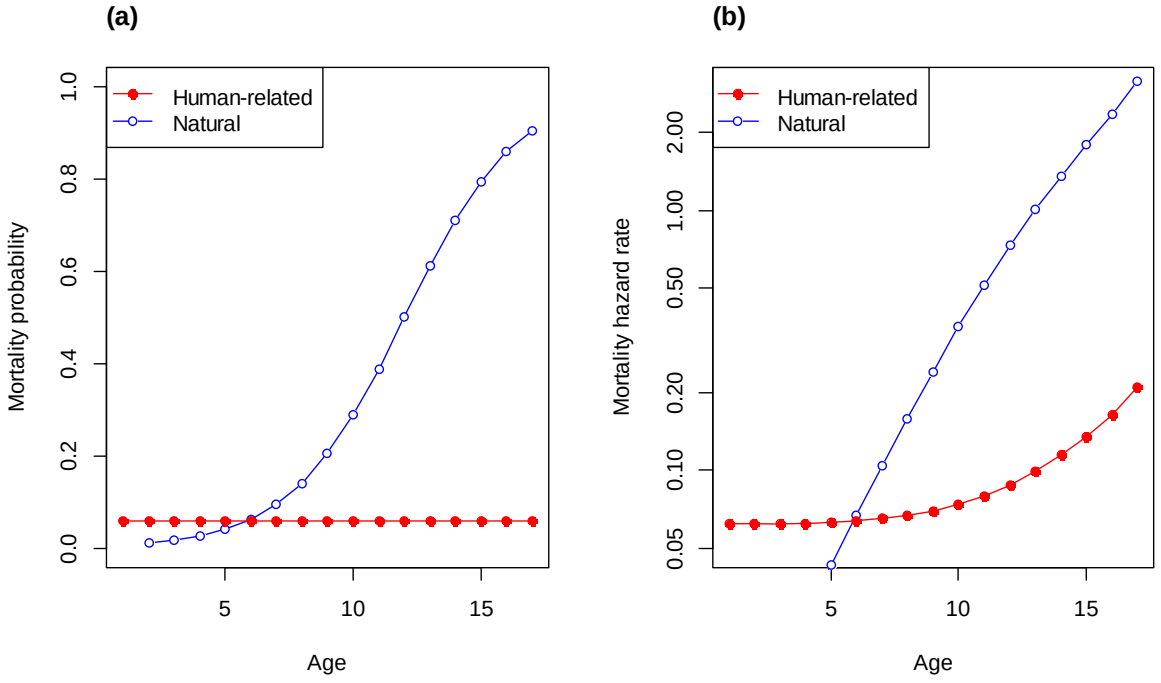

Figure 4. An example of two age related competing causes of mortality; human-related and non-human-related (natural). Mortality probabilities are plotted in panel a, while corresponding time-averaged mortality hazard rates are shown in panel b. The fact that one of the two mortality probabilities is constant (red line in panel a) while the other increases with age, implies that both cause specific mortality hazard rates must increase with age (panel b) – see equations in Table 1. Mortality hazard rates (panel b) are plotted on a logarithmic scale for easier comparison of relative differences. In this example, the mortality hazard rate for human related causes (red line in panel b) increases more than 3 times from age 1 to age 17 even though mortality probability remains constant. Compare figure with Figure 4 in Koons *et al.* (2014).

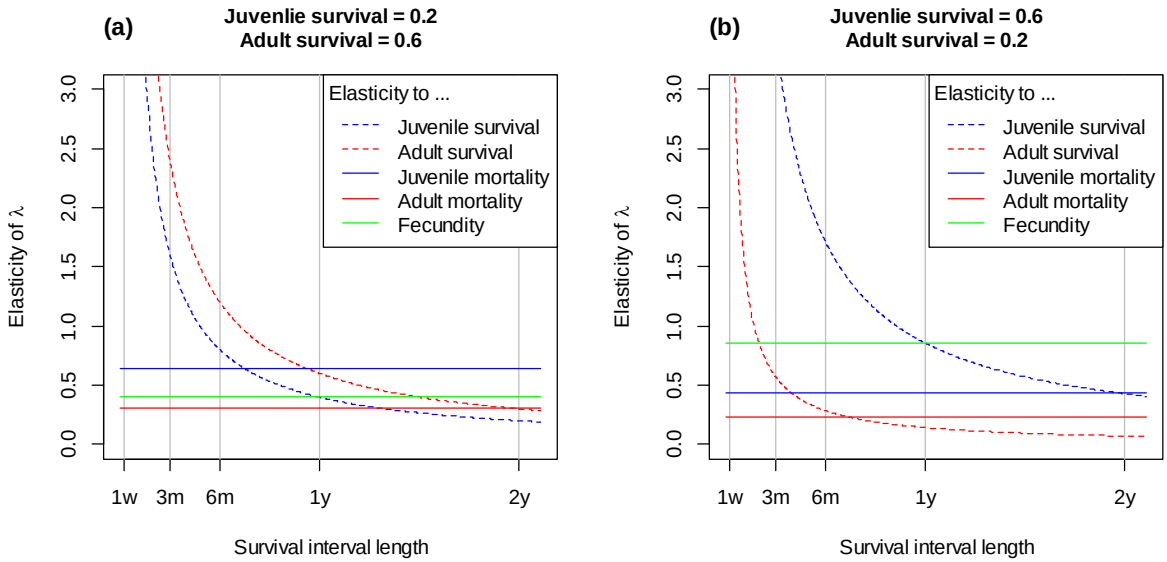

Figure 5. Elasticity of population growth rate (λ) to juvenile (blue lines) and adult (red lines) survival (dashed lines) and mortality hazard rate (solid lines), as well as to fecundity (green lines), when the model is parameterized with survival probabilities over different interval lengths (x-axis). The model assumes maturation at age 1 years and fecundity is 2 offspring per individuals for all ages. Vertical lines indicate 1 week, 3 months, 6 months, 1 year and 2 years. In panel (a), yearly juvenile survival is 0.2, while adult survival is 0.6. In panel (b), yearly juvenile survival is 0.6, while adult survival is 0.2. For mortality hazard rates, the negative elasticity (i.e., the elasticity of lambda to a *decrease* in mortality hazard rate) is plotted for easier comparison.

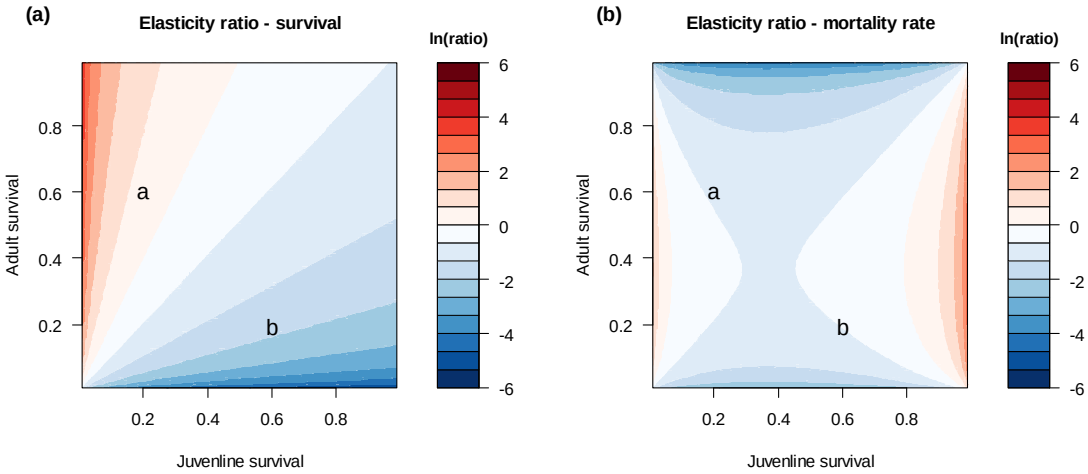

Figure 6. Elasticity of population growth rate (λ) to adult survival (panel a) and mortality hazard rate (panel *b*) relative to elasticity to respectively juvenile survival and juvenile mortality rate. In both panels, the elasticity ratios (colour-key on a $\log_e$-scale) are shown for all possible combinations of juvenile (x-axis) and adult survival (y-axis). As in Figure 5, maturation occurs at age 1 and fecundity is 2 offspring per individuals for all ages. Plotted labels "a" and "b" correspond to the survival probabilities used in respectively panel (a) and panel (b) of Figure 5. It can be shown that the elasticity ratio with respect to mortality (panel b) is the hazard ratio $\left(\frac{\bar{h}_{adult}}{\bar{h}_{juvenile}}\right)$ times the elasticity ratio with respect to survival (panel a).

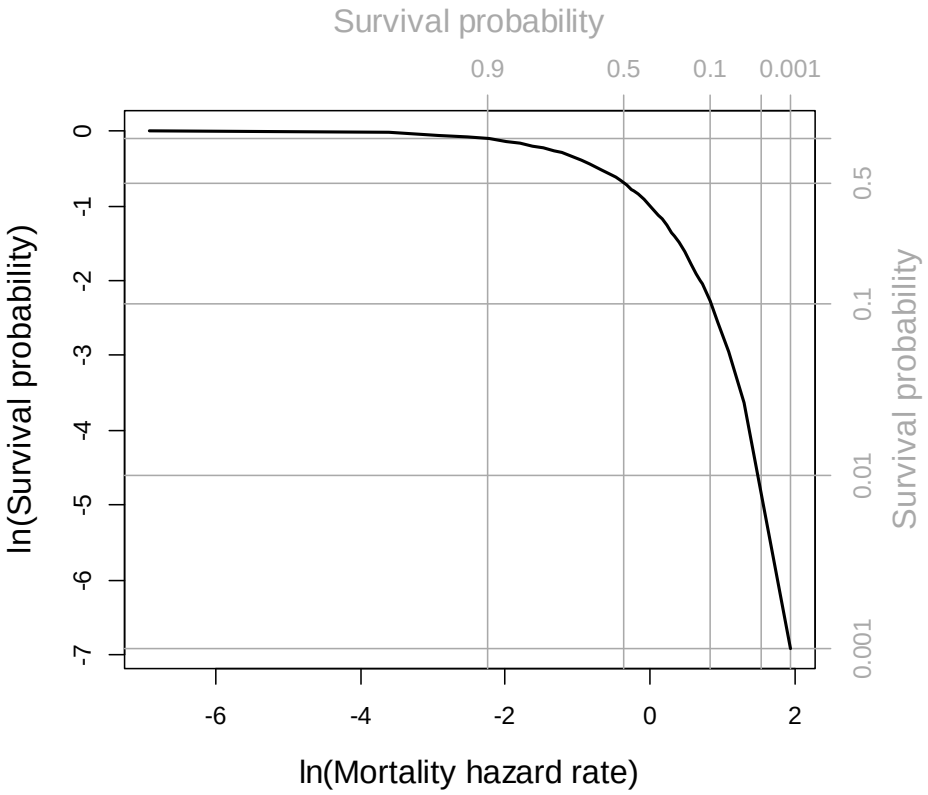

Figure 7. The natural logarithm of survival probability (y-axis) as a function of the natural logarithm of time-averaged mortality hazard rate (x-axis). The slope of the curve is the relative change in survival per relative change in mortality hazard rate. Corresponding survival probabilities are shown in the top and right axes and grey lines.

Table 1. Matrix of three alternative parameterizations of state transition probabilities in a multi-state model of two mutually exclusive and collectively exhaustive causes of mortality, assuming that cause specific hazard rates remain proportional within intervals. Definitions of the parameters are given in the diagonal cells. These parameters may be interval and individual specific, and may be modelled as random effects or as functions of covariates in a hierarchical model.

Parameterization	**State transition probabilities**[1]	**Survival and proportion died of cause 1**	**Mortality hazard rates**
State transition probabilities; $P_1 + P_2 + S = 1$ (Schaub & Lebreton 2004; Bischof *et al.* 2009; Servanty *et al.* 2010; Péron 2013; Koons *et al.* 2014)	$\boldsymbol{P_1}$ **= Probability of dying of cause 1** $\boldsymbol{P_2}$ **= Probability of dying of cause 2** $\boldsymbol{S = 1 - P_1 - P_2}$ **= Survival probability**	$S = 1 - P_1 - P_2$ $\alpha = \frac{P_1}{P_1 + P_2}$	$\bar{h}_1 = -\log(1 - P_1 - P_2)\frac{P_1}{P_1 + P_2}$ $\bar{h}_2 = -\log(1 - P_1 - P_2)\frac{P_2}{P_1 + P_2}$
Survival and proportion died of cause 1; S and α (Schaub & Pradel 2004; Schaub 2009; Souchay & Schaub 2016)	$P_1 = (1 - S)\alpha$ $P_2 = (1 - S)(1 - \alpha)$ $S = S$	$\boldsymbol{S}$ **= Survival probability** $\boldsymbol{\alpha}$ **= Proportion died of cause 1**[2]	$\bar{h}_1 = -\log(S)\,\alpha$ $\bar{h}_2 = -\log(S)\,(1 - \alpha)$
Mortality hazard rates; h_1 and h_2 (Box S1 - this paper)	$P_1 = \left(1 - e^{-(\bar{h}_1 + \bar{h}_2)}\right)\frac{\bar{h}_1}{\bar{h}_1 + \bar{h}_2}$ $P_1 = \left(1 - e^{-(\bar{h}_1 + \bar{h}_2)}\right)\frac{\bar{h}_2}{\bar{h}_1 + \bar{h}_2}$ $S = e^{-(\bar{h}_1 + \bar{h}_2)}$	$S = e^{-(\bar{h}_1 + \bar{h}_2)}$ $\alpha = \frac{\bar{h}_1}{\bar{h}_1 + \bar{h}_2}$	$\boldsymbol{\bar{h}_1}$ **= Time-averaged mortality hazard rate of cause 1** $\boldsymbol{\bar{h}_2}$ **= Time-averaged mortality hazard rate of cause 2**

[1] Parameters P_1, P_2 and S constrained to sum to 1 by e.g. the 'mlogit' link-function ($P_k = \frac{\exp(\eta_k)}{1 + \sum_{l=1}^{K} \exp(\eta_l)}$ and $S = 1 - \sum_{k=1}^{K} P_k$).

[2] May also be interpreted as proportion of total hazard rate attributable to cause 1 when cause specific hazard rates are proportional within intervals, but not necessarily constant (see equation (3)).

Glossary

Mortality hazard rate: The latent intensity of deadly events that an individual is exposed to (Box 1). Synonymous to 'force of mortality' and 'failure rate'. Often referred to as just 'mortality rate'.

Survival probability: The probability that an individual survives an interval of a given length given that it is alive at the beginning of the interval. A unit-free measurement on an **absolute scale**. Sometimes misleadingly referred to as 'survival rate'.

Mortality probability: The probability that an individual dies during a given time interval, equal to one minus the survival probability for the same interval. Often misleadingly referred to as 'mortality rate'.

Risk: Usually synonymous to **mortality probability**.

Odds: When applied to survival, **survival probability** divided by **mortality probability**.

Odds-ratio: One **odds** divided by another.

Hazard ratio: One **hazard rate** divided by another.

Logit-link: A link-function on the form log('odds') = 'linear predictor' $= \beta_0 + \beta_1 x_1 + \beta_2 x_2 + \cdots = \boldsymbol{\beta x}$.

Loglog-link: A link-function on the form log('time-averaged mortality hazard rate') = 'linear predictor' (see Box 1).

Absolute-scaled variable: In measurement theory (Houle *et al.* 2011), a variable on a scale type with no permissible transformations, meaning that there are no transformations that preserve the relevant relationships among measurements (e.g. probabilities).

Ratio-scaled variable: In measurement theory (Houle *et al.* 2011; Collett 2014), a positive variable with a true (nature-given) zero (zero refers to "non-existence" or "total absence"). Examples include age, mass and mortality hazard rates. The only permissible transformations that preserve the relevant relationships among measurements are multiplications and divisions by strictly positive numbers.

Absolute difference/effect: A difference or effect quantified as one quantity minus another. E.g., the absolute difference between mortality rate values 2 and 1 is 1.

Relative difference/effect: A difference or effect quantified as one quantity divided by another. E.g., the relative difference between mortality rate values 2 and 1 is 2. The **absolute difference** on a logarithmic scale equals the logarithm of the relative difference since $\log(a) - \log(b) = \log(a/b)$.

Linear contrast: The **absolute difference** between two predicted values at the scale of the link-function. E.g. one log('odds') value minus another when using a **logit-link**, or the absolute difference between two values of log('time-averaged mortality hazard rate') when using a **loglog-link** on **survival probability**.

Log-linear models: Models on the form log('prediction') $= \beta_0 + \beta_1 x_1 + \beta_2 x_2 + \cdots$. When the x's are log-transformed values of a predictor variable, a **relative difference** in the predictor variable leads to a **relative difference** in the predictions (e.g., a 10% increase in the predictor variable x_1 leads to a relative difference in the prediction equal to 1.1^{β_1}).

Frailty: Unobserved individual heterogeneity in mortality hazard rates. In the presence of **frailty**, the population will be increasingly dominated by individuals with low **mortality hazard rates** at higher age-classes. Hence, age-related patterns in population level mortality rates are not representative of individual level patterns.

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Supporting Information for "Study mortality with hazard rates, not probabilities"

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Box S1 – Multiple sources of mortality and competing risks

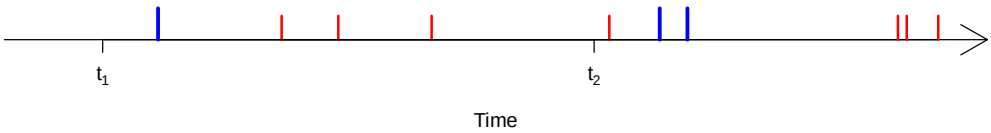

Figure S1. Realized times of two types of independent recurrent events (or "deadly events experienced by an imortal individual")

Figure S1 illustrates a situation where another ("blue") cause of mortality has been added to the "red" cause of mortality illustrated in Fig. B1 (Box 1 in the main text). If we, for simplicity, assume that the hazard rates for these two causes of mortality are constant over an interval with values h_1 and h_2, the total mortality hazard rate is now $h_1 + h_2$, and the probability of surviving the interval of length Δt is $S = e^{-(h_1+h_2)\Delta t}$. We could have divided each of these causes of mortality into several mutually exclusive sub-categories, but survival probability would have remained the same.

It is essential to realize that adding the blue cause of mortality in this example reduces the probability of dying from the red cause, even though the hazard rate representing the red cause remains the same. This is due to the fact that the individual now may die from a blue cause before a red event takes place. The probability of dying from a specific cause (often referred to as a "**risk**"), when the hazard rate of this cause remains unchanged, will always go down when the hazard rate of other mortality causes increase (or go up if the hazard rate of other causes decrease). One may say that the risks of death are competing, and the probabilities of dying from different causes are intrinsically linked. Hence, a change in the probability of dying of a specific cause may be due to changes in any of the cause specific mortality hazard rates. When studying *e.g.*, environmental influence on different causes of mortality, or how different causes of mortality vary and co-vary among groups of individuals or among years within a population, it is therefore of prime interest to estimate the hazard rates of different causes of mortality rather than the probabilities of dying from different causes.

Discrete-time models (such as classical capture recapture models) that incorporate different causes of mortality involve state-transition probability parameters representing the probabilities that an individual that is alive at one time-point (t_1) is either alive or dead from specific causes at a later point in time (t_2). These transition probabilities should sum to 1. Hence, $S + \sum_{k=1}^{K} P_k = 1$, where S is the survival probability and the $P_k{}'s$ are the probabilities of dying from different causes classified in K categories. Assuming, for simplicity, that each of the cause specific hazard rates, h_k are constant

within intervals, we know from above that $S = e^{-(\sum_{k=1}^{K} h_k)\Delta t}$. To find the probability of dying from cause k, P_k, we may use the definition of hazard rates as

$$h(t) = \lim_{dt \to 0} \frac{\Pr(t \leq T < t + dt | t < T)}{dt} \tag{1}$$

(Collett 2014) where T is time of death and dt is an infinitesimally small unit of time (i.e., $h(t)dt$ is the probability that the individual will die before time $t + dt$ given that it is alive at time t). From this we see that the relationship between the hazard rate and the probability density for time of death at time t, $f(t)$, is

$$h(t) = \frac{f(t)}{S(t)} \tag{2}$$

where $S(t)$ is the probability of being alive at time t, which equals $1 - \int_0^t f(t)dt$. Using this relationship, we can now find the probability density function for the time of death from cause k, $f_k(\tau)$, where $\tau = t - t_1$ is the time since the beginning of the interval,

$$f_k(\tau) = h_k e^{-\tau \sum_{l=1}^{K} h_l} \tag{3}$$

Note that this density function is not a not proper probability distributions function as it does not integrate to 1 over infinite time. Instead, it integrates to the probability of ever dying from cause k, which in the case of constant hazard rates becomes $h_k / \sum_{k=1}^{K} h_k$. To find the probability of dying from cause k before the end of the interval, we need to integrate $f_k(\tau)$ from $\tau = 0$ to $\tau = \Delta t$ $(= t_2 - t_1)$, and get

$$P_k = \int_0^{\Delta t} f_k(\tau) d\tau = \left(1 - e^{-\Delta t \sum_{l=1}^{K} h_l}\right) \frac{h_k}{\sum_{l=1}^{K} h_l} = (1 - S) \frac{h_k}{\sum_{l=1}^{K} h_l} \tag{4}$$

This expression can be generalized to cases where cause specific hazard rates may vary over time but remain proportional throughout the interval such that $\alpha_k = h_k(t) / \sum_{l=1}^{K} h_l\,(t)$, in which case we get

$$P_k = (1 - S)\alpha_k \tag{5}$$

Note that the conditional probability of having died from cause k, given that the individual has died, in the case of proportional hazard rates is the fraction of the total mortality hazard rate attributed to the specific cause, α_k, which in this case equals the probability of ever dying of this cause. This is, however, not the case when hazard rates are not proportional over time.

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